Critical Care

RESEARCH Open Access

Vasopressin use across shock states: international insights from an international ESICM-endorsed survey: the PRESS Survey

Mathieu Jozwiak[1,2*], Vladimir L. Cousin[3], Daniel De Backer[4], Manu L. N. G. Malbrain[5,6], Xavier Monnet[7], Antonio Messina[8,9] and Michelle S. Chew[10] on behalf of the Cardiovascular Dynamics section of the European Society of Intensive Care Medicine

Abstract

Background Vasopressin is currently suggested as a second-line vasopressor in patients with septic shock and persistent arterial hypotension. This survey aimed to assess the current use of vasopressin and terlipressin in patients with septic shock.

Methods An international survey endorsed by the European Society of the Intensive Care Medicine. All items addressed the different areas of uncertainty and research identified from current international recommendations.

Results 1919 intensivists from 124 countries responded to the survey: 1220(64%) worked in Europe, 1430(74%) in high and upper-middle income level countries and 916(48%) in academic ICUs. Vasopressin or terlipressin were available to 1830(95%) respondents. The main reported indications were refractory septic shock, catecholamine sparing in all types of refractory shock, refractory vasoplegic shock (other than septic) without reduced left ventricular systolic function and septic shock with high cardiac output and low systemic vascular resistance (73%,53%,51% and 49% of respondents, respectively). The main reported contraindications were non-occlusive mesenteric ischemia and digital or skin ischemia. Overall, 1506(78%) respondents used vasopressin or terlipressin, 93% of them as a second-line vasopressor after initial resuscitation, mainly to increase blood pressure and reduce the catecholamine load. The dose of norepinephrine alone or the duration of norepinephrine administration alone triggered vasopressin and terlipressin initiation for 98% and 53% of respondents, respectively: the most frequent dose of norepinephrine base was 0.25–0.50 µg/kg/min and the most frequent duration was > 2-6 h. The combination of the dose of norepinephrine base and the duration of its administration triggered vasopressin and terlipressin initiation for 78% of respondents, the most frequent combination being a norepinephrine base dose > 0.25 or > 0.50 µg/kg/min for > 2-6 h. An initial dose of vasopressin of 0.01 or 0.03 U/min was used by 50% of respondents. The mean arterial pressure level triggered the tapering of vasopressin or terlipressin for 88% of respondents. Discontinuation started after the dose of the first-line vasopressor had been lowered below a predefined threshold for 43% of respondents and was progressive for 89% of respondents.

*Correspondence:
Mathieu Jozwiak
jozwiak.m@chu-nice.fr

Full list of author information is available at the end of the article

© The Author(s) 2025. **Open Access** This article is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License, which permits any non-commercial use, sharing, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if you modified the licensed material. You do not have permission under this licence to share adapted material derived from this article or parts of it. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit http://creativecommons.org/licenses/by-nc-nd/4.0/.

SHOUT
SHARE
IT

Conclusions Vasopressin is commonly used to treat patients with septic shock, but its handling (doses and discontinuation practices) remains heterogeneous, indicating the need for guidance in its clinical use.

Keywords Septic shock, Vasopressors, Norepinephrine, Decatecholaminization, Vasopressor load, Vasopressin, Terlipressin

Background

Septic shock is one of the leading causes of admission in the intensive care unit (ICU) and is still associated with high mortality and morbidity [1]. The initial hemodynamic management of patients with septic shock consists of fluid resuscitation and norepinephrine administration, which is currently the recommended first-line vasopressor [1].

Current guidelines suggest adding vasopressin rather than increasing the norepinephrine dose when mean arterial pressure (MAP) is considered inadequate despite doses of norepinephrine of 0.25–0.5 µg/kg/min [1]. Introduction of vasopressin is justified by vasopressin deficiency occurring in one third of patients with septic shock [2, 3] and using vasopressin in patients with septic shock already receiving norepinephrine may be associated with decreased requirement of renal replacement therapy and less atrial fibrillation [4, 5]. Additionally, using high-doses of norepinephrine may be associated with poorer outcomes [6–8] and adverse cardiac effects like myocardial inflammation and necrosis [9]. On the other hand, vasopressin use is associated with a higher incidence of digital ischemia, without any effect on mortality [4, 5].

Supporting evidence underpinning current guidelines is limited and many questions regarding the use of vasopressin and its analogues remain unresolved: their availability, the triggers for their initiation and the timing for their initiation. This international survey aimed to address the use of vasopressin and its analogues in patients with septic shock in ICUs around the world.

Methods

The survey was supported by the European Society of Intensive Care Medicine (ESICM) and utilized a snowball sampling approach, enabling participants to share the survey link via social media and email distribution lists. The survey was conducted in English language only and comprised 34 questions (Supplementary material) divided into five main sections: (1) "Respondents and ICU characteristics", (2) "Vasopressors use", (3) "Vasopressin and terlipressin use", (4) "Administration of vasopressin or terlipressin in patients with septic shock" and (5) "Continuation-discontinuation of vasopressin or terlipressin in patients with septic shock". The fourth and fifth sections of the survey were reserved solely for intensivists who use vasopressin or terlipressin.

Survey development

Survey questions were developed iteratively (MJ and VC) according to the different areas of uncertainty and research identified from current international recommendations [1]. Initially, MJ and VC independently identified relevant questions from the current guidelines and compiled a comprehensive list. Redundant items were then eliminated, and the remaining questions were reviewed collaboratively. Only those with full agreement between the two authors were retained. To ensure validity, the final set of questions was further reviewed by a steering committee (DB, XM, AM and MC). Lastly, the survey was tested by members of the ESICM cardiovascular dynamics section, with adjustments made based on their feedback.

Survey dissemination and population

The survey was distributed electronically with SurveyMonkey Inc. (www.surveymonkey.com). To account for potential differences in individual practice, responses were collected at the intensivist level rather than as a single ICU-wide submission. No personal data was collected, and participation was anonymous with no login required. To enhance completion rates and maintain internal consistency, respondents received an alert before final submission, prompting them to address any unanswered mandatory questions. Once submitted, responses could not be reviewed or modified. The survey was promoted on the ESICM website and remained open from September 2023 to July 2024. All ESICM members were invited via email, with the survey link sent to their registered addresses in September 2023, followed by reminder emails in December 2023, February 2024, and May 2024. Additionally, the survey was widely shared across ESICM's social media platforms. The survey was also endorsed by the International Fluid Academy (IFA, www.fluidacademy.org) and promoted on the IFA members portal and email reminders and social media channels.

Survey reporting

The survey's methodology and results are reported following the CHERRIES (Checklist for Reporting Results of Internet E-Surveys) guidelines [10]. Ethical approval was not required due to the voluntary participation of respondents and the absence of any individual patient data collection.

Statistical analysis

Results were expressed as numbers (percentage) and were reported according to the status of ICUs (academic *vs.* general) in which the respondents worked. The income level of countries was defined according to the World Bank classification. Missing data were displayed with item denominators. Descriptive statistics were only carried out on the available data. Subgroup analyses were performed using Chi-2 test or Fisher exact test with appropriate number of degrees of freedom as appropriate. A generalized mixed model with three random effects (continent, World Bank classification and academic status of ICU) and hierarchical data analysis was also performed to address the issue of practice heterogeneity for the following three variables: (i) dose of norepinephrine base as a trigger for vasopressin and terlipressin administration, (ii) duration of norepinephrine administration as a trigger for vasopressin and terlipressin administration and (iii), combination of the dose of norepinephrine base and the duration of administration as a trigger for vasopressin and terlipressin administration. All tests were two-sided and a p-value < 0.05 was considered statistically significant. All statistical analyses were performed using EasyMedStat® (version 3.40; www.easymedstat.com) and R (version 3.6.3, R foundation for Statistical Computing Vienna, Austria).

Results

Respondents

Overall, 1919 intensivists from 124 different countries responded to the survey: 1220 (64%) respondents worked in Europe (Fig. 1), 1430 (74%) in high income level countries, 916 (48%) worked in academic ICUs and 1016 (53%) had > 10 years of experience in ICU (Table 1). The characteristics of respondents and ICUs according to continents, income level of countries and academic status of ICUs are summarized in Tables S1, S2 and S3 respectively.

The hemodynamic monitoring most widely used in patients receiving vasopressors was echocardiography (84% of respondents) (Table 2). Echocardiography remained the most widely used hemodynamic monitoring, regardless of continents (Table S4), income level of countries (Table S5) and academic status of ICUs (Table S6).

Use of vasopressors

A local protocol for vasopressors was available to 1157 (60%) respondents. Norepinephrine base was used by 1083 (56%) respondents (Table 2) and was more likely to be used in academic than in non-academic ICUs (60 *vs.* 54%, p = 0.01) (Table S6). Compared to norepinephrine tartrate, norepinephrine base was more likely to be

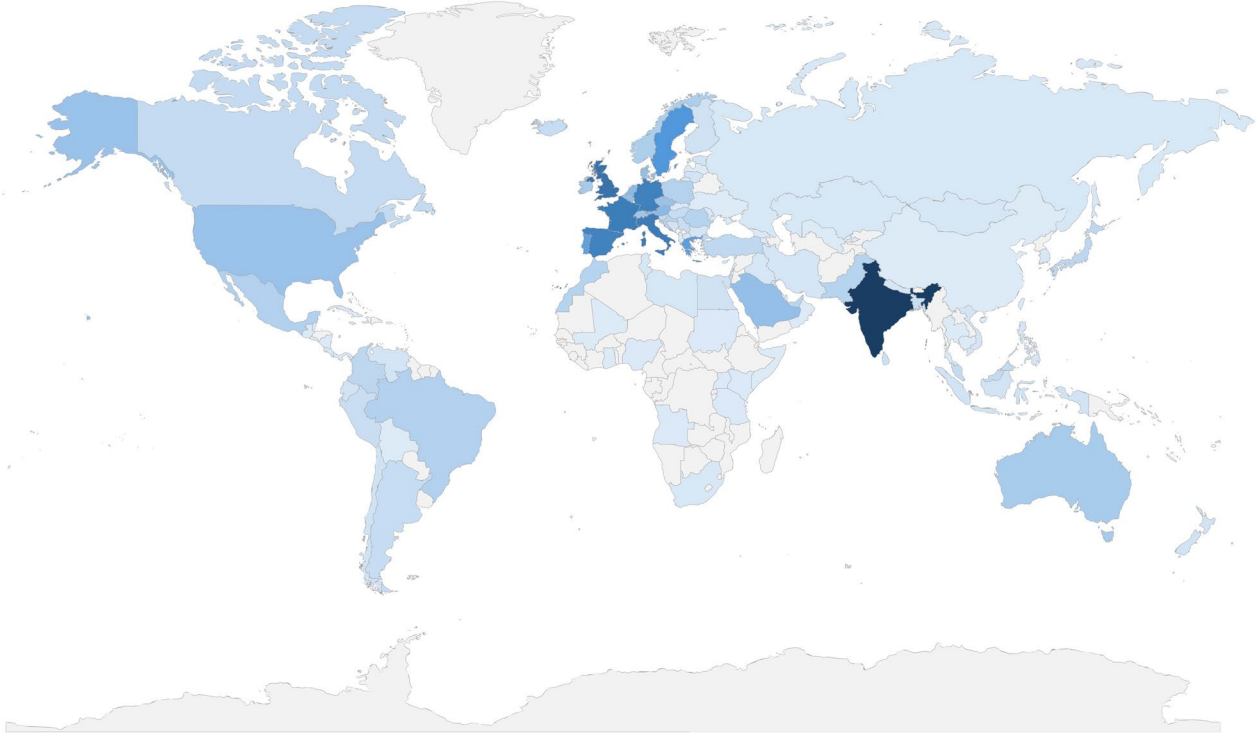

Fig. 1 World map showing the number of respondents per country. The intensity of the blue color indicates the number of respondents per country: the lightest blue color indicates countries with the lowest number of respondents (Luxembourg, Ukraine, Latvia, Cyprus, Mali, Ghana, Somalia, Democratic Republic of Congo, Bolivia, El Salvador, Guatemala, Cambodia, Thailand, Armenia, Uzbekistan, China, Lebanon, Oman, Belize and Samoa, n = 1) and the darkest blue color indicates the country with the highest number of respondents (India, n = 163)

Table 1 Respondents and ICU characteristics

	Respondents n=1919
Continent	
Europe	1220 (64%)
North America	93 (5%)
South America	91 (5%)
Africa	58 (3%)
Asia	419 (21%)
Oceania	38 (2%)
Income level of countries according to the World Bank classification	
High-income level	1430 (74%)
Upper-middle income level	211 (11%)
Lower-middle income level	271 (14%)
Low-income level	7 (1%)
Type of ICU	
Medical ICU	723 (38%)
Surgical ICU	296 (15%)
Medical-Surgical ICU	764 (40%)
Cardiac surgery	117 (6%)
Neurological ICU	13 (<1%)
Burn Unit	6 (<1%)
Number of beds	
>20	637 (33%)
16-20	306 (16%)
11-15	448 (23%)
5-10	486 (26%)
<5	42 (2%)
Number of patients admitted per year	
>2000	242 (12%)
1501-2000	204 (11%)
1001-1500	399 (21%)
500-1000	689 (36%)
<500	380 (20%)
Experience as intensivist (in years)	
>15	657 (34%)
10-15	359 (19%)
5-10	370 (19%)
2-5	289 (15%)
<2	112 (6%)
Not specialized yet, in training	132 (7%)
Main specialty	
Intensivist	1238 (65%)
Anesthesiologist	487 (25%)
Internal medicine	119 (6%)
Others	75 (4%)

ICU: intensive care unit

used in all continents except in Asia (50 vs. 50%, p = 0.94) (Table S4, Figure S1) and in all countries except lower-middle income countries (43 *vs.* 57%, p < 0.001) (Table S5, Figure S2). Mean arterial pressure was the main target of vasopressor use (Table 2, Table S4, Table S5 and Table S6).

Corticosteroids were administered by 1260 (66%) respondents before considering a second-line vasopressor (Table 2). Corticosteroids were less likely to be used in North America compared to other continents (Table S4) but their administration did not differ according to income level of countries (Table S5).

The main reported potential indications for vasopressin and terlipressin were refractory septic shock (73% of respondents), catecholamine sparing in all types of refractory shock (53% of respondents), refractory vasoplegic shock (other than septic) without reduced left ventricular systolic function (51% of respondents) and septic shock with high cardiac output and low systemic vascular resistance (SVR) (49% of respondents) (Table 2, Fig. 2). The two main reported potential contraindications were non-occlusive mesenteric ischemia and digital or skin ischemia for vasopressin (40% of respondents for both) and terlipressin (35% and 36% of respondents, respectively). For 16% of respondents, there was no contraindication to vasopressin and terlipressin (Table 2). The most feared adverse effects were mesenteric ischemia, digital or skin ischemia and cardiac ischemia (67%, 60% and 30% of respondents respectively) (Table 2, Fig. 3). Potential indications and contraindications as well as the most feared adverse effects of vasopressin and terlipressin according to continents, income level of countries and academic status of ICUs are summarized in Tables S4, S5 and S6 respectively.

Use of vasopressin and terlipressin

Vasopressin or terlipressin were available to 1830 (95%) respondents and were used, when available, by 1506 (78%) respondents (Table 2). vasopressin and terlipressin were mainly used as second-line vasopressor after initial resuscitation with fluids and norepinephrine (93% of respondents) and vasopressin was the most widely used agent (96% of respondents) (Table 3). When vasopressin and terlipressin were contraindicated, 712 (47%) respondents used epinephrine and 422 (28%) accepted lower blood pressure levels when tissue perfusion variables were acceptable (Table 3).

The two main goals for using vasopressin and terlipressin were to increase blood pressure and reduce the catecholamine load (85% and 72% of respondents respectively) (Table 3). The two main triggers for vasopressin and terlipressin initiation were low mean arterial pressure and low SVR (87% and 46% of respondents respectively) (Table 3, Fig. 4).

The dose of norepinephrine alone was considered as a trigger for vasopressin and terlipressin initiation by 1470 (98%) respondents. In that case, a norepinephrine base dose between 0.25 and 0.50 µg/kg/min was the most frequent trigger (48% of respondents) (Table 3, Fig. 5A). The proportion of respondents who considered

Table 2 Use of vasopressors in patients with septic shock

	Respondents n=1919
Local protocol for vasopressors	1157 (60%)
Type of norepinephrine used	
Base	1083 (56%)
Tartrate	836 (44%)
Vasopressin or terlipressin available	1830 (95%)
Use of vasopressin and terlipressin when available	1506 (78%)
Corticosteroids administration before considering a second-line vasopressor	1260 (66%)
Targets for vasopressors	
Mean arterial pressure	1839 (96%)
Peripheral perfusion variables	916 (48%)
Urine output	984 (51%)
Biological target	1112 (58%)
None	6 (<1%)
Hemodynamic monitoring other than arterial pressure in patients receiving vasopressors	
Echocardiography	1615 (84%)
Pulmonary artery catheter	426 (22%)
Transpulmonary thermodilution	874 (45%)
Uncalibrated pulse contour analysis	599 (31%)
None	129 (7%)
Potential indications for vasopressin or terlipressin	
Septic shock refractory to initial resuscitation	1401 (73%)
Septic shock with high risk of renal failure	406 (21%)
Septic shock with oliguria or anuria	428 (22%)
Septic shock with atrial arrhythmia	361 (19%)
Septic shock with high CO and low systemic vascular resistance	951 (50%)
Septic shock with RV dysfunction	428 (22%)
Septic shock in patient with ARDS	482 (25%)
Septic shock in patient with cirrhosis	625 (33%)
Refractory vasoplegic shock without LV systolic dysfunction	980 (51%)
Refractory vasoplegic shock with LV systolic dysfunction	546 (28%)
Refractory vasoplegic shock with RV dysfunction	563 (29%)
Catecholamine sparing in all types of refractory shock	1014 (53%)
Other indications	36 (2%)
Potential contraindications of vasopressin	
History of gastrointestinal vasculopathy	>413 (22%)
History of unstable coronary syndrome	596 (31%)
History of systemic sclerosis	206 (11%)
History of vasospastic diseases	681 (36%)
History of peripheral vasculopathy	429 (22%)
Digital or skin ischemia	773 (40%)
Non-occlusive mesenteric ischemia	771 (40%)
Postoperative period with vascular or bowel anastomosis	392 (20%)
RV failure with severe PH	259 (12%)
Allergy	7 (<1%)
Others	25 (1%)
No absolute contraindication	30 (2%)
None	332 (17%)
Potential contraindications of terlipressin	
History of gastrointestinal vasculopathy	381 (20%)
History of unstable coronary syndrome	558 (29%)
History of systemic sclerosis	213 (11%)
History of vasospastic diseases	587 (31%)

Table 2 (continued)

	Respondents n=1919
History of peripheral vasculopathy	450 (23%)
Digital or skin ischemia	686 (36%)
Non-occlusive mesenteric ischemia	670 (35%)
Postoperative period with vascular or bowel anastomosis	370 (19%)
RV failure with severe PH	230 (12%)
Allergy	3 (<1%)
Others	13 (<1%)
No absolute contraindication	12 (<1%)
None	321 (17%)
Feared adverse effects of vasopressin and terlipressin	
Digital or skin ischemia	1157 (60%)
Mesenteric ischemia	1294 (67%)
Cardiac arrhythmias	202 (10%)
Cardiac ischemia	569 (30%)
Decrease in CO	412 (21%)
Worsening of existing severe PH and RV failure	406 (21%)
Hyponatremia	193 (10%)
Others	6 (<1%)
None	71 (4%)

ARDS: acute respiratory distress syndrome; CO: cardiac output; LV: left ventricular; PH: pulmonary hypertension; RV: right ventricular

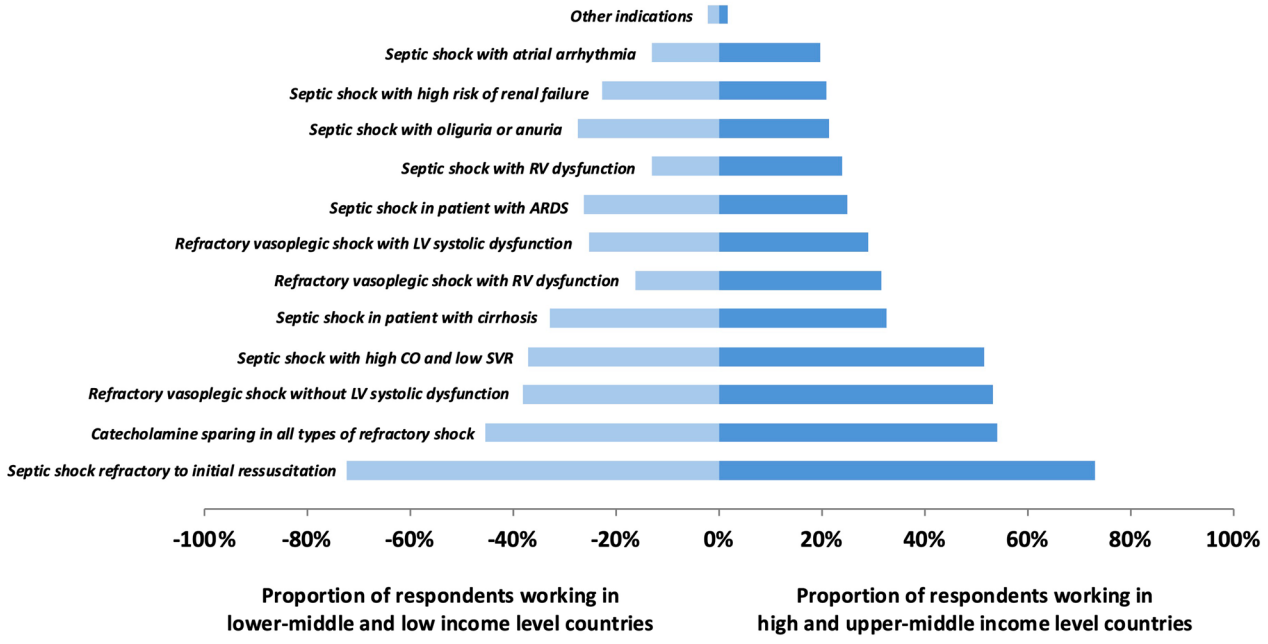
Other indications
Septic shock with atrial arrhythmia
Septic shock with high risk of renal failure
Septic shock with oliguria or anuria
Septic shock with RV dysfunction
Septic shock in patient with ARDS
Refractory vasoplegic shock with LV systolic dysfunction
Refractory vasoplegic shock with RV dysfunction
Septic shock in patient with cirrhosis
Septic shock with high CO and low SVR
Refractory vasoplegic shock without LV systolic dysfunction
Catecholamine sparing in all types of refractory shock
Septic shock refractory to initial ressuscitation
-100%
-80%
-60%
-40%
-20%
0%
20%
40%
60%
80%
100%
Proportion of respondents working in lower-middle and low income level countries
Proportion of respondents working in high and upper-middle income level countries

Fig. 2 Indications for vasopressin or terlipressin use. Bar graphs showing the indications for vasopressin or terlipressin use in respondents working in high and upper-middle income level countries (dark blue bar graphs) and in lower-middle and low-income level countries (light blue bar graphs). LV: left ventricular, CO: cardiac output, SVR: systemic vascular resistance, RV: right ventricular, ARDS: acute respiratory distress syndrome

a norepinephrine base dose between 0.25 and 0.50 µg/kg/min to be a trigger did not differ between those with a local protocol for vasopressors compared to those without (47% *vs.* 49%, p = 0.43). The duration of norepinephrine administration alone was considered as a trigger for vasopressin and terlipressin initiation by 801 (53%) respondents. In that case, a duration of administration for > 2–6 h was the most frequent trigger (26% of respondents) (Table 3, Fig. 5B). The proportion of respondents who considered a duration of administration for > 2–6 h to be a trigger did not differ between those with a local protocol for vasopressors compared to those without (26% *vs.* 27%, p = 0.73). The combination of the dose of norepinephrine base and the duration of

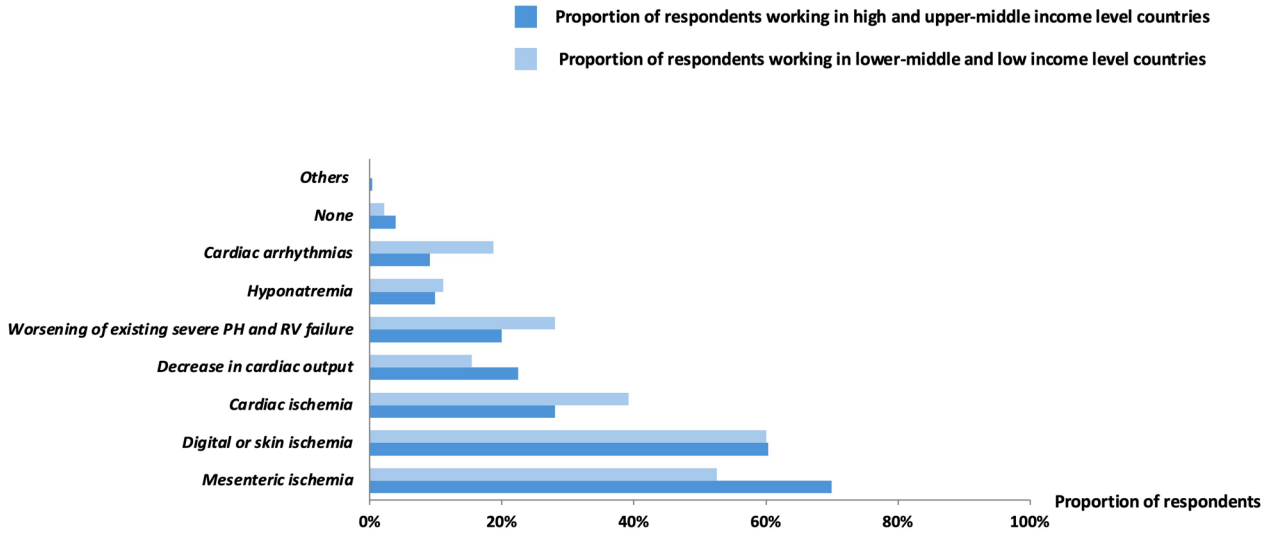

Fig. 3 Potential feared adverse effects of vasopressin and terlipressin. Bar graphs showing the potential feared adverse effects of vasopressin and terlipressin in respondents working in high and upper-middle income level countries (dark blue bar graphs) and in lower-middle and low-income level countries (light blue bar graphs). PH: pulmonary hypertension, RV: right ventricular

its administration was considered as a trigger for vasopressin and terlipressin initiation by 1173 (78%) respondents. In that case, a norepinephrine base dose > 0.25 or > 0.50 µg/kg/min for > 2–6 h was the most frequent trigger (53% of respondents) (Table 3, Fig. 5C). The use of vasopressin and terlipressin according to continents, income level of countries and academic status of ICUs are summarized in Tables S7, S8 and S9 respectively. Type of ICU and experience as intensivist were both associated with heterogeneity in practice regarding the combination of the dose of norepinephrine base and duration of administration as a trigger for vasopressin and terlipressin administration (Tables S10, S11 and S12). In contrast there was no association with continent, World Bank classification of income level and academic status of ICU (Tables S10, S11 and S12).

Doses of vasopressin and terlipressin

For vasopressin, the most frequent initial doses were 0.01 or 0.03 U/min (28% and 23% of respondents, respectively) (Table 4, Fig. 6A) and the most frequent maximum dose was 0.04 U/min (36% of respondents) (Table 4, Fig. 6B). For terlipressin, the most frequent initial doses were 2 and 4 mg/day and the most frequent maximum dose of terlipressin was 4 mg/day. A fixed dose was considered by 656 (46%) respondents, 650 (46%) titrated the initial dose to mean arterial pressure and 111 (8%) titrated the initial dose to SVR (Table 4, Fig. 6C). Doses of vasopressin and terlipressin according to continents, income level of countries and academic status of ICUs are summarized in Tables S13, S14 and S15 respectively.

Discontinuation of vasopressin and terlipressin

The main triggers for tapering vasopressin and terlipressin were the mean arterial pressure level (88% of respondents) and the first-line vasopressor dose (46% of respondents). Vasopressin and terlipressin were tapered first by 31% of respondents, at the same time as first line vasopressor by 18% of respondents and after the dose of the first-line vasopressor had been lowered below a predefined threshold by 43% of respondents. A progressive decrease in doses of vasopressin and terlipressin was considered by 89% of respondents (Table 4). Discontinuation of vasopressin and terlipressin according to continents, income level of countries and academic status of ICUs are summarized in Tables S13, S14 and S15 respectively.

Discussion

The role of vasopressin and terlipressin, a synthetic analogue of vasopressin, in the management of patients with septic shock is still debated. This survey highlights that vasopressin is commonly used among patients with septic shock as a second-line vasopressor, with its introduction mostly based on a dose of norepinephrine base < 0.5 µg/kg/min, without paying attention to timing under vasopressors in half of the cases. Also, renal dysfunction and atrial fibrillation, which have been suggested to be improved by vasopressin [4, 5] are considered as indication by less than 20% of responders.

Norepinephrine exists as norepinephrine base and norepinephrine tartrate. Both formulations, which are to date not available everywhere, are not equivalent with base being twice as potent as norepinephrine tartrate (2 mg norepinephrine tartrate = 1 mg norepinephrine base) [11]. Norepinephrine base was more frequently

Table 3 Use of vasopressin and terlipressin in patients with septic shock

	Respondents n=1506
Use of vasopressin and terlipressin	
Vasopressin	1441 (96%)
Terlipressin	37 (2%)
Vasopressin or Terlipressin	16 (1%)
Vasopressin and Terlipressin	12 (1%)
Goals and triggers for vasopressin and terlipressin initiation	
Goals	
To improve cardiac output	346 (23%)
To improve blood pressure	1277 (85%)
To improve urine output	584 (39%)
To reduce administration of intravenous fluid	608 (40%)
To reduce risk of renal replacement therapy	380 (25%)
To reduce risk of cardiac arrhythmias	307 (20%)
To decrease the catecholamine load	1099 (73%)
Clinical and biological triggers	
Abnormal peripheral perfusion	422 (28%)
Oliguria	377 (25%)
Tachycardia	199 (13%)
Persistent tachycardic atrial fibrillation	158 (10%)
Low systolic arterial pressure	377 (25%)
Low mean arterial pressure	1313 (87%)
Low diastolic arterial pressure	451 (30%)
Low systemic vascular resistance	699 (46%)
Slow lactate clearance	380 (25%)
High PCO_2gap (>6 mmHg)	165 (11%)
Low S(c)vO_2	189 (12%)
None	36 (2%)
Dose of norepinephrine base as a trigger	
0.15-0.25 µg/kg/min	203 (13%)
0.25-0.50 µg/kg/min	718 (48%)
0.50-1.00 µg/kg/min	370 (25%)
1-2 µg/kg/min	127 (8%)
>2 µg/kg/min	52 (4%)
Not a triggering factor	36 (2%)
Duration of norepinephrine administration as a trigger	
<2h	230 (15%)
2-6h	395 (26%)
6-12h	96 (6%)
>12h	80 (5%)
Not a triggering factor	705 (48%)
Combination of the dose of norepinephrine base and the duration of administration as a trigger	
>0.15 µg/kg/min for 2-6 hours	82 (5%)
>0.25 µg/kg/min for 2-6 hours	464 (31%)
>0.5 µg/kg/min for 2-6 hours	334 (22%)
>1 µg/kg/min for 2-6 hours	90 (6%)
>2 µg/kg/min for 2-6 hours	39 (3%)
>0.15 µg/kg/min for >6 hours	9 (<1%)
>0.25 µg/kg/min for >6 hours	47 (3%)
>0.5 µg/kg/min for >6 hours	54 (4%)
>1 µg/kg/min for >6hours	32 (2%)
>2 µg/kg/min for >6 hours	22 (1%)
Not a triggering factor	333 (22%)

Table 3 (continued)

	Respondents n=1506
Timing for vasopressin and terlipressin use	
Phase of resuscitation	
First-line vasopressor	4 (<1%)
Second-line vasopressor, after initial resuscitation	1407 (93%)
Third-line vasopressor, as rescue therapy	107 (6%)
First-line vasopressor	
Norepinephrine	1493 (99%)
Epinephrine	3 (<1%)
Vasopressin	4 (<1%)
Terlipressin	0 (0%)
Angiotensin II	0 (0%)
Phenylephrine	2 (<1%)
Methylene blue	0 (0%)
Dopamine	4 (<1%)
Second-line vasopressor	
Norepinephrine	12 (1%)
Epinephrine	65 (4%)
Vasopressin	1365 (91%)
Terlipressin	26 (2%)
Vasopressin or Terlipressin	16 (<1%)
Angiotensin II	2 (<1%)
Phenylephrine	3 (<1%)
Methylene blue	2 (<1%)
Dopamine	14 (<1%)
I do not use second-line vasopressor	1 (<1%)
Third-line vasopressor	
Norepinephrine	0 (0%)
Epinephrine	750 (50%)
Vasopressin	84 (6%)
Terlipressin	23 (1%)
Angiotensin II	64 (4%)
Phenylephrine	32 (2%)
Methylene blue	201 (13%)
Dopamine	41 (3%)
No third-line vasopressor	310 (21%)
Vasopressor used when vasopressin and terlipressin are contraindicated	
Epinephrine	712 (47%)
Angiotensin II	69 (5%)
Phenylephrine	43 (3%)
Methylene blue	196 (13%)
Dopamine	64 (4%)
Accept lower blood pressure when tissue perfusion variables are acceptable	422 (28%)

PCO_2: partial pressure of carbon dioxide; $S(c)vO_2$: (central) venous oxygen saturation

used than norepinephrine tartrate, with heterogeneity according to continents, income level of countries and academic status of ICUs. This heterogeneity results in inconsistency in the definition of refractory shock [12] and between studies for comparing patients' outcomes according to the vasopressor load [11]. To prevent confusion, we requested the responders to report whether they used base or tartrate and converted the dose trigger into base equivalents for homogeneity. A recent consensus paper therefore suggests that norepinephrine base should be adopted as the reference for norepinephrine dosing and formulations in ICUs [13].

Vasopressin and terlipressin are widely available worldwide and vasopressin were used by most respondents. Although, the use of terlipressin in patients with septic shock is not currently recommended by the Surviving

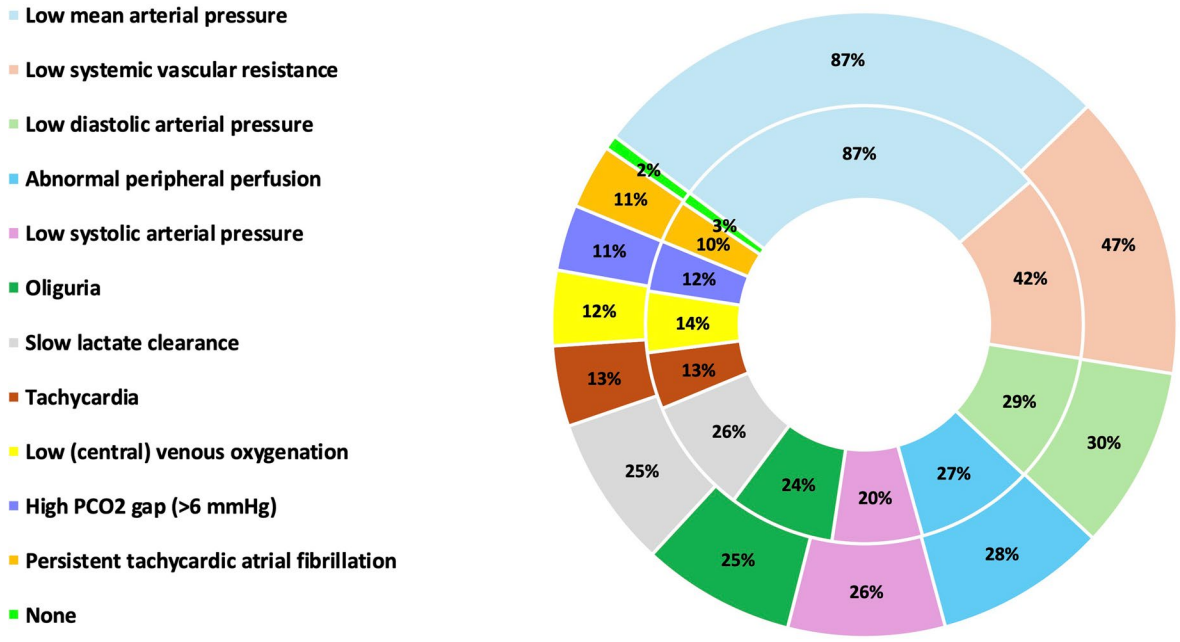

Fig. 4 Clinical and biological triggers for vasopressin or terlipressin initiation. Clinical and biological triggers for vasopressin or terlipressin initiation in respondents working in high and upper-middle income level countries (outer ring) and lower-middle and low-income level countries (inner ring). The proportion of respondents who report using the trigger is indicated for each trigger. SVR: systemic vascular resistance, PCO_2: partial pressure of carbon dioxide

Sepsis Campaign guidelines [1], its use might be on particular interest in subgroups of patients with septic shock and cirrhosis [14] or acute-on-chronic liver failure [15], as terlipressin is recommended for the management of variceal bleeding due to its effect on portal venous pressure [16]. Their main indication was refractory septic shock, and the main goals of their use were to increase blood pressure and reduce the catecholamine load. Such a strategy, sometimes called "decatecholaminization" [17, 18] aims to decrease the norepinephrine dose and limit the adrenergic side effects when treating sepsis-induced vasoplegia as well as to bypass downregulation and reduced sensitivity of adrenergic-receptors [19]. Two large randomized trials showed that administering vasopressin in addition to norepinephrine in patients with septic shock allowed the dose of norepinephrine and thus the vasopressor load to be decreased and the administration of norepinephrine to be discontinued earlier [20, 21]. Such a strategy could also have a fluid-sparing effect, limiting fluid overload, which is associated with poor outcomes in patients with septic shock [22]. Only 66% of respondents administered corticosteroids before considering a second-line vasopressor, even though it has been shown that corticosteroids administration can also reduce the vasopressor load and hasten the resolution of shock [23, 24]. Moreover, a *post hoc* analysis of the VASST study found an interaction between corticosteroids and vasopressin administration, as the combination of vasopressin and corticosteroids was associated with lower mortality [25]. Vasopressin and terlipressin were mainly used as second-line vasopressor after initial resuscitation with fluids and norepinephrine. In practice, 98% of respondents considered the norepinephrine dose alone to be a trigger for the initiation of vasopressin and terlipressin, with a norepinephrine base dose between 0.25 and 0.50 µg/kg/min, in agreement with current international recommendations [1]. Only 78% of respondents considered the combination of the dose of norepinephrine base and the duration of its administration to be a trigger for the initiation of vasopressin and terlipressin, with a norepinephrine base dose >0.25 or >0.50 µg/kg/min for >2-6h. It must be noted that we did not consider a duration of norepinephrine administration <2h in our survey, which may have influenced the way the results of triggers for the initiation of vasopressin and terlipressin were reported. Nevertheless, the timing of second-line vasopressor initiation remains an unresolved issue [26, 27]. Some studies found an association between a low dose of norepinephrine at the time of vasopressin administration and better patient outcomes [20, 28, 29] while other studies found an association between a short time from the onset of septic shock to vasopressin administration and better patient outcomes [30, 31]. Meta-analyses showed that the administration of vasopressin within the first six hours after the onset of septic shock was associated with a shorter duration of vasopressor use and a lower risk of renal replacement therapy [32] and that early administration of vasopressors in patients with septic shock may be

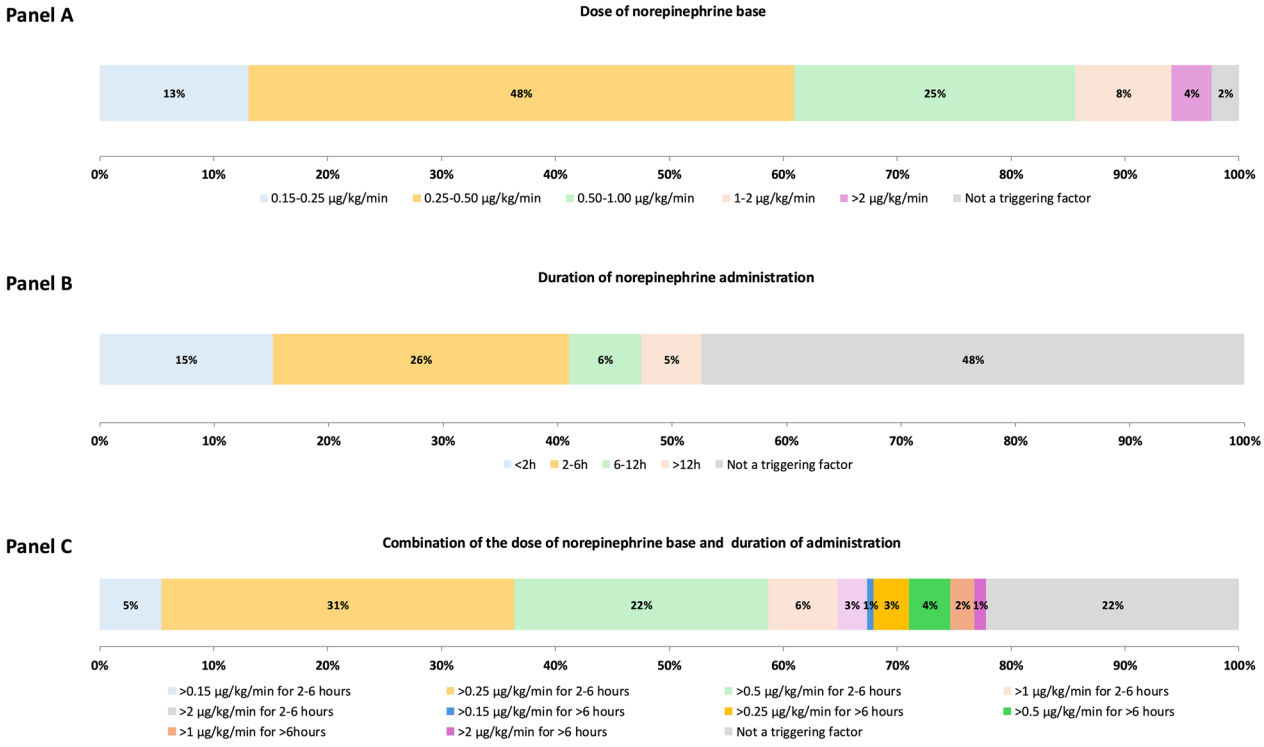

Fig. 5 Dose of norepinephrine base and duration of norepinephrine administration considered as triggers for vasopressin or terlipressin initiation. *Panel A*: dose of norepinephrine base considered to be a trigger for vasopressin or terlipressin initiation in all respondents using them in patients with septic shock. Each bar shows the distribution (in percent) of respondents for each norepinephrine dose range. *Panel B*: duration of norepinephrine administration considered to be a trigger for vasopressin or terlipressin initiation in all respondents using them in patients with septic shock. Each bar shows the distribution (in percent) of respondents for each range of duration of norepinephrine administration. *Panel C*: combination of the dose of norepinephrine base and duration of administration considered to be a trigger for vasopressin or terlipressin initiation in all respondents using them in patients with septic shock. Each bar shows the distribution (in percent) of respondents for each combination range

associated with lower mortality [33]. Two recent observational trials using modern approaches to minimize biases, may support the earlier use of vasopressin both in terms of norepinephrine dosages and duration of administration [34, 35]. In the OVISS trial, the authors showed that using artificial intelligence with a generated reinforcement learning model to optimize the vasopressin initiation rule compared to intensivist's practice, would have led to earlier vasopressin administration at lower concurrent doses of norepinephrine in more patients, while observing a lower mortality if vasopressin was initiated according to the model rule [34]. In a multicenter target trial emulation, vasopressin administration within the first 6 hours of septic shock was associated with lower mortality if commenced at lower norepinephrine doses (<0.25 µg/kg/min), i.e. lower than those recommended by the Surviving Sepsis Campaign guidelines [35]. Collectively, these results suggest that it may be relevant to also take into account both the norepinephrine dose and the duration of its administration before considering a second-line non-adrenergic vasopressor. According to the protocol used in the VASST and VANISH studies [20, 21], the most frequent initial doses of vasopressin ranged from 0.01 to 0.03 U/min with a maximum dose of 0.06 U/min. Similarly, the most frequent initial dose of terlipressin ranged from 2 to 4 mg/d with a maximum dose of 4 mg/d, in line with a previous multicentric trial on terlipressin in patients with septic shock [36]. We found significant heterogeneity in the way doses of vasopressin and terlipressin were adjusted with less than half of respondents following protocols indicated in the randomized studies, in which the dose of vasopressin and terlipressin was titrated according to mean arterial pressure [20, 21]. Vasopressin and terlipressin were most often tapered after the dose of the first-line vasopressor had been lowered below a predefined threshold with a progressive decrease in doses. To date, the question of their discontinuation remains unresolved with retrospective studies showing that norepinephrine should be stopped first to avoid arterial hypotension [37], while small randomized trials found the opposite [38, 39]. From a physiological point of view, it makes sense to stop vasopressin first, since vasopressin is a pure vasopressor [19], unlike norepinephrine, which is an inopressor [40]. Discontinuing norepinephrine first could induce or unmask left ventricular systolic dysfunction due to the combination of

Table 4 Doses and discontinuation of vasopressin and terlipressin in patients with septic shock

	Respondents n=1417
Doses of vasopressin and terlipressin	
Initial dose	
0.01 U/min	393 (28%)
0.02 U/min	290 (20%)
0.03 U/min	321 (23%)
0.04 U/min	297 (21%)
0.06 U/min	40 (3%)
0.0005 U/kg/min	19 (1%)
0.001 U/kg/min	7 (1%)
Other initial doses	17 (1%)
Use of terlipressin	33 (2%)
Adjustment of the dose	
Fixed dose same as initial dose	434 (31%)
Fixed dose at 0.03 U/min	104 (7%)
Fixed dose at 0.04 U/min	66 (5%)
Fixed dose at 0.06 U/min	23 (1%)
Other fixed doses in U/min	21 (1%)
Fixed dose of terlipressin	8 (1%)
Titration on mean arterial pressure	650 (46%)
Titration on systemic vascular resistance	111 (8%)
Maximum dose	
0.03 U/min	339 (24%)
0.04 U/min	515 (36%)
0.06 U/min	315 (22%)
0.001 U/kg/min	15 (1%)
0.01 U/kg/min	19 (1%)
0.05 U/kg/min	38 (3%)
Other maximum doses	71 (5%)
Maximum dose of terlipressin	31 (2%)
No maximum dose	69 (5%)
Discontinuation of vasopressin and terlipressin	
Triggers for tapering	
Mean arterial pressure	1245 (88%)
Peripheral perfusion variables	613 (43%)
Urine output	460 (32%)
Dose of the first-line vasopressor	650 (46%)
Systemic vascular resistance	447 (31%)
Biological variables (lactate, PCO_2 gap, $S(c)vO_2$…)	474 (34%)
Secondary effects of vasopressin or analogues	377 (27%)
None	4 (<1%)
Timing of tapering in vasopressin and terlipression	
Vasopressin or analogues are tapered first	442 (31%)
At the same time as first-line vasopressor	114 (8%)
After the first-line vasopressor	254 (18%)
Below a predefined threshold dose of first-line vasopressor	607 (43%)
Modalities of tapering in vasopressin and terlipressin	
Progressive decrease in doses	1261 (89%)
Abrupt discontinuation	156 (11%)

PCO_2: partial pressure of carbon dioxide; $S(c)vO_2$: (central) venous oxygen saturation

increased left ventricular afterload and loss of norepinephrine inotropic activity, especially in patients with impaired cardiac function, due to their heart's dependency on afterload.

Our survey has some limitations. First, this was a voluntary survey conducted in English language only. Consequently, the risk of selection and response bias cannot be ruled out, as only physicians with some understanding of English with an interest in vasopressin use participated, which could influence the representativeness of the results. Second, respondents were asked about their experience in ICU but were not directly about their professional role, which may have affected the accuracy of some responses. Third, there is as yet no universally accepted definition of refractory shock, which may have influenced survey responses, as respondents' interpretation may have varied. Fourth, we could not calculate the response rate. Fifth, some centers may have been overrepresented, as one response per intensivist was expected. Sixth, we did not collect additional data regarding the vasopressor protocols in use. Consequently, we were unable to determine whether variations existed between different protocols or if discrepancies arose from deviations in adherence, which could account for the heterogeneity observed. Finally, it must be kept in mind that the results of this survey only represent the declarated answers of the respondents, which may potentially be more theoretical than practical, and that the responses in this survey may therefore differ somewhat from the clinical practice. However, this is the largest international survey on vasopressin and terlipressin use to date, providing a comprehensive and reliable overview of current practices in patients with septic shock, highlighting substantial heterogeneity between intensivists. The knowledge gaps from this study will inform future research directions, including randomized controlled trials or observational registries to better understand the efficacy and effects of vasopressors in shock.

Conclusions

This large and international survey showed that vasopressin was commonly used to treat patients with septic shock, but its handling (doses and discontinuation practices) remains heterogeneous. This survey therefore suggests room for improvement moving toward a more personalized use of vasopressin instead of implementing vasopressin when a fixed dose of norepinephrine is reached.

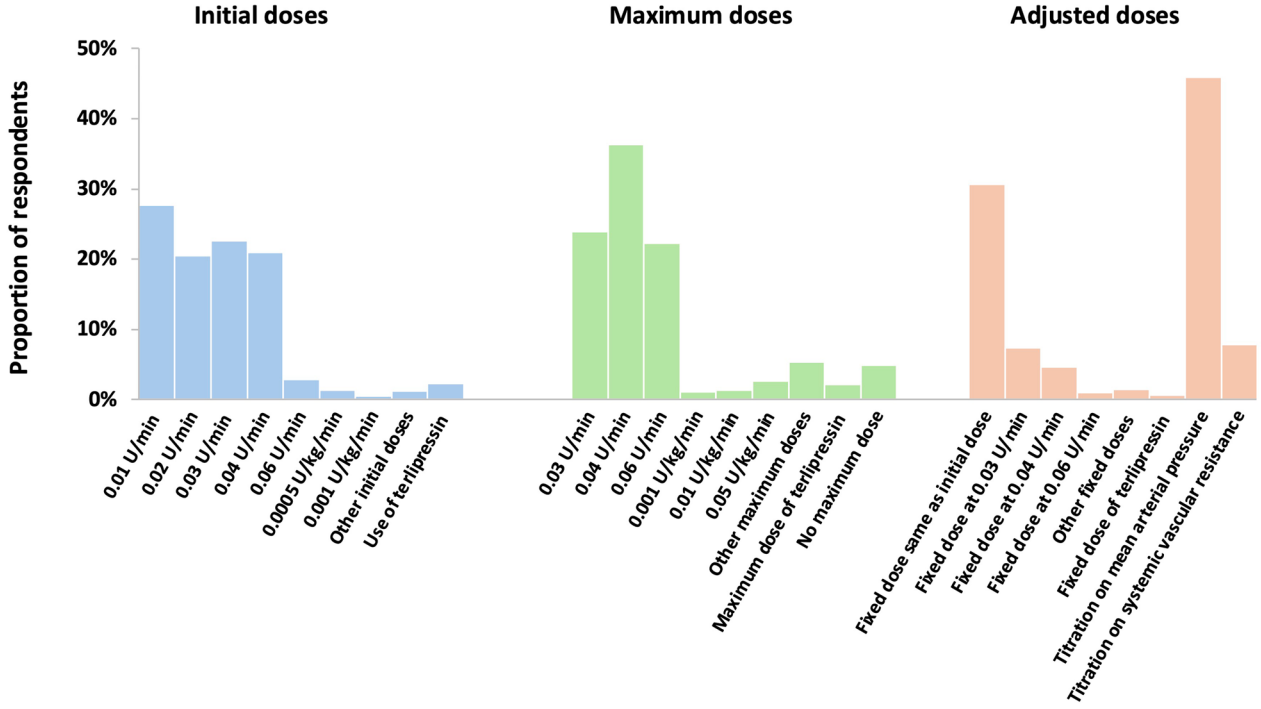

Fig. 6 Dose management of vasopressin and terlipressin. Bar graphs showing dose management of vasopressin and terlipressin in all respondents using them in patients with septic shock. The left panel represents the initial dose (blue bars), the middle panel indicates the maximum doses (green bars) and the right panel indicates the adjusted doses (orange panel)

List of abbreviations

ICU Intensive care unit
SVR Systemic vascular resistance

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13054-025-05505-5.

Additional file 1

Acknowledgements
None.

Author contributions
MJ and VC conceived and designed the survey. MJ, VC, DDB, MM, XM, AM and MC analyzed and interpreted the data. MJ drafted the first version of the manuscript report. All authors contributed drafting the manuscript and approved the final version of the manuscript.

Funding
No funding to declare.

Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
The survey was endorsed by the European Society of Intensive Care Medicine (ESICM).

Consent for publication
Not applicable.

Competing interests
XM received fees for giving lectures from Getinge, Baxter healthcare, BD, Masimo and AOP health. XM received fees for consulting from Getinge and BD. XM received unrestricted research grants from Baxter healthcare and Masimo. MLNGM is Professor of Critical Care Research at the 1 st Department of Anaesthesiology and Intensive Therapy, Medical University of Lublin, Poland. He is co-founder, past-President and current Treasurer of WSACS (The Abdominal Compartment Society, http://www.wsacs.org). He is member of the medical advisory Board of Pulsion Medical Systems (now fully part of Getinge group), Sentinel Medical, and Baxter. He consults for BBraun, Becton Dickinson, Fresenius Kabi, Lexin, Maltron, Spiegelberg, Medtronic, MedCaptain, and Holtech Medical, and received speaker's fees from PeerVoice, WICCEM and Fresenius-Kabi. He holds stock options for Sentinel, Serenno and Potrero. He is co-founder and President of the International Fluid Academy (IFA). The IFA (http://www.fluidacademy.org) is integrated within the not-for-profit charitable organization iMERiT, International Medical Education and Research Initiative, under Belgian law. MC has received speaker's and advisory board honoraria from Edwards Lifesciences, AOP Health, Laboratorie Agguetant and Philips Healthcare. The other authors have no conflict of interest to declare.

Author details
[1]Service de Médecine Intensive Réanimation, CHU de Nice, 151 Route Saint Antoine de Ginestière, 06200 Nice, France
[2]UR2CA, Equipe CARRES Physiologie Cardio-Respiratoire, Université Côte d'Azur, Nice, France
[3]Service de Réanimation Pédiatrique, Hôpital de Bicêtre, Université Paris-Saclay, AP-HP, Le Kremlin-Bicêtre, France
[4]Department of Intensive Care Medicine, CHIREC Hospitals, Université Libre de Bruxelles, Brussels, Belgium
[5]First Department of Anaesthesiology and Intensive Therapy, Medical University of Lublin, Lublin, Poland
[6]International Fluid Academy, Lovenjoel, Belgium
[7]Service de Médecine Intensive-Réanimation, Hôpital de Bicêtre, DMU CORREVE, Inserm UMR S_999, IHU PROMETHEUS, FHU SEPSIS, Groupe de Recherche Clinique CARMAS, Université Paris-Saclay, AP-HP, Le Kremlin-Bicêtre, France

[8]IRCCS Humanitas Research Hospital, Via Manzoni 56, 20089 Rozzano, Mialan, Italy
[9]Department of Biomedical Sciences, Humanitas University, Via Levi Montalcini 4, Pieve Emanuele, Milan, Italy
[10]Department of Perioperative Medicine and Intensive Care, Karolinska University Hospital, Huddinge, Sweden

Received: 29 April 2025 / Accepted: 14 June 2025
Published online: 03 July 2025

References

1. Evans L, Rhodes A, Alhazzani W, Antonelli M, Coopersmith CM, French C, et al. Surviving sepsis campaign: international guidelines for management of sepsis and septic shock 2021. Intensive Care Med. 2021;47(11):1181–247.
2. Landry DW, Levin HR, Gallant EM, Ashton RC Jr, Seo S, D'Alessandro D, et al. Vasopressin deficiency contributes to the vasodilation of septic shock. Circulation. 1997;95(5):1122–5.
3. Sharshar T, Blanchard A, Paillard M, Raphael JC, Gajdos P, Annane D. Circulating vasopressin levels in septic shock. Crit Care Med. 2003;31(6):1752–8.
4. McIntyre WF, Um KJ, Alhazzani W, Lengyel AP, Hajjar L, Gordon AC, et al. Association of Vasopressin Plus Catecholamine Vasopressors vs Catecholamines Alone With Atrial Fibrillation in Patients With Distributive Shock: A Systematic Review and Meta-analysis. JAMA. 2018;319(18):1889–900.
5. Nagendran M, Russell JA, Walley KR, Brett SJ, Perkins GD, Hajjar L, et al. Vasopressin in septic shock: an individual patient data meta-analysis of randomised controlled trials. Intensive Care Med. 2019;45(6):844–55.
6. Dunser MW, Ruokonen E, Pettila V, Ulmer H, Torgersen C, Schmittinger CA, et al. Association of arterial blood pressure and vasopressor load with septic shock mortality: a post hoc analysis of a multicenter trial. Crit Care. 2009;13(6):R181.
7. Auchet T, Regnier MA, Girerd N, Levy B. Outcome of patients with septic shock and high-dose vasopressor therapy. Ann Intensive Care. 2017;7(1):43.
8. Sato R, Duggal A, Sacha GL, Rudoni MA, Yataco AC, Khanna AK, et al. The Relationship Between Norepinephrine Equivalent Dose of Vasopressors Within 24 Hours From the Onset of Septic Shock and In-Hospital Mortality Rate. Chest. 2023;163(1):148–51.
9. Schmittinger CA, Dunser MW, Torgersen C, Luckner G, Lorenz I, Schmid S, et al. Histologic pathologies of the myocardium in septic shock: a prospective observational study. Shock. 2013;39(4):329–35.
10. Eysenbach G. Improving the quality of Web surveys: the Checklist for Reporting Results of Internet E-Surveys (CHERRIES). J Med Internet Res. 2004;6(3):e34.
11. Leone M, Goyer I, Levy B, Dunser MW, Asfar P, Jentzer JC. Dose of norepinephrine: the devil is in the details. Intensive Care Med. 2022;48(5):638–40.
12. Bakker J, Kattan E, Annane D, Castro R, Cecconi M, De Backer D, et al. Current practice and evolving concepts in septic shock resuscitation. Intensive Care Med. 2022;48(2):148–63.
13. Wieruszewski PM, Leone M, Kaas-Hansen BS, Dugar S, Legrand M, McKenzie CA, et al. Position Paper on the Reporting of Norepinephrine Formulations in Critical Care from the Society of Critical Care Medicine and European Society of Intensive Care Medicine Joint Task Force. Crit Care Med. 2024;52(4):521–30.
14. Choudhury A, Kedarisetty CK, Vashishtha C, Saini D, Kumar S, Maiwall R, et al. A randomized trial comparing terlipressin and noradrenaline in patients with cirrhosis and septic shock. Liver Int. 2017;37(4):552–61.
15. Gupta T, Saini A, Gaur V, Goel A. Comparative Study of Terlipressin and Noradrenaline as Vasopressors in Patients With Acute-on-chronic Liver Failure and Septic Shock: A Randomized Controlled Trial. J Clin Exp Hepatol. 2025;15(3): 102494.
16. Skvarce J, Bui A, Oro P, Sachar S, Harnegie MP, Kapoor A, et al. Multisystem hemodynamic effects of terlipressin in cirrhosis: A scoping review. J Crit Care. 2025;87: 155038.
17. Lesur O, Delile E, Asfar P, Radermacher P. Hemodynamic support in the early phase of septic shock: a review of challenges and unanswered questions. Ann Intensive Care. 2018;8(1):102.
18. Demiselle J, Fage N, Radermacher P, Asfar P. Vasopressin and its analogues in shock states: a review. Ann Intensive Care. 2020;10(1):9.
19. Russell JA. Vasopressor therapy in critically ill patients with shock. Intensive Care Med. 2019;45(11):1503–17.
20. Russell JA, Walley KR, Singer J, Gordon AC, Hebert PC, Cooper DJ, et al. Vasopressin versus norepinephrine infusion in patients with septic shock. N Engl J Med. 2008;358(9):877–87.
21. Gordon AC, Mason AJ, Thirunavukkarasu N, Perkins GD, Cecconi M, Cepkova M, et al. Effect of Early Vasopressin vs Norepinephrine on Kidney Failure in Patients With Septic Shock: The VANISH Randomized Clinical Trial. JAMA. 2016;316(5):509–18.
22. Mekontso Dessap A, AlShamsi F, Belletti A, De Backer D, Delaney A, Moller MH, et al. European Society of Intensive Care Medicine (ESICM) 2025 clinical practice guideline on fluid therapy in adult critically ill patients: part 2-the volume of resuscitation fluids. Intensive Care Med. 2025;51(3):461–77.
23. Annane D, Renault A, Brun-Buisson C, Megarbane B, Quenot JP, Siami S, et al. Hydrocortisone plus Fludrocortisone for Adults with Septic Shock. N Engl J Med. 2018;378(9):809–18.
24. Venkatesh B, Finfer S, Cohen J, Rajbhandari D, Arabi Y, Bellomo R, et al. Adjunctive Glucocorticoid Therapy in Patients with Septic Shock. N Engl J Med. 2018;378(9):797–808.
25. Russell JA, Walley KR, Gordon AC, Cooper DJ, Hébert PC, Singer J, et al. Interaction of vasopressin infusion, corticosteroid treatment, and mortality of septic shock. Crit Care Med. 2009;37(3):811–8.
26. Scheeren TWL, Bakker J, De Backer D, Annane D, Asfar P, Boerma EC, et al. Current use of vasopressors in septic shock. Ann Intensive Care. 2019;9(1):20.
27. De Backer D, Hajjar L, Monnet X. Vasoconstriction in septic shock. Intensive Care Med. 2024;50(3):459–62.
28. Sacha GL, Lam SW, Wang L, Duggal A, Reddy AJ, Bauer SR. Association of Catecholamine Dose, Lactate, and Shock Duration at Vasopressin Initiation With Mortality in Patients With Septic Shock. Crit Care Med. 2022;50(4):614–23.
29. Xu J, Cai H, Zheng X. Timing of vasopressin initiation and mortality in patients with septic shock: analysis of the MIMIC-III and MIMIC-IV databases. BMC Infect Dis. 2023;23(1):199.
30. Rydz AC, Elefritz JL, Conroy M, Disney KA, Miller CJ, Porter K, et al. Early Initiation of Vasopressin Reduces Organ Failure and Mortality in Septic Shock. Shock. 2022;58(4):269–74.
31. White KC, Costa-Pinto R, Chaba A, McIlroy P, Senthuran S, Luke S, et al. Timing of adjunctive vasopressin initiation for septic shock patients and hospital mortality: A multicentre observational study. Crit Care Resusc. 2024;26(4):295–302.
32. Ye E, Ye H, Wang S, Fang X. Initiation Timing of Vasopressor in Patients with Septic Shock: A Systematic Review and Meta-Analysis. Shock. 2023;60(5):627–36.
33. Ma CH, Healy J, Kinteh E, Ma CC, Tzeng CT, Chou EH, et al. Extremely early initiation of vasopressors might not decrease short-term mortality for adults with septic shock: a systematic review and meta-analysis. Ann Intensive Care. 2025;15(1):18.
34. Kalimouttou A, Kennedy JN, Feng J, Singh H, Saria S, Angus DC, et al. Optimal Vasopressin Initiation in Septic Shock: The OVISS Reinforcement Learning Study. Jama. 2025.
35. White KC, Costa-Pinto R, Blank S, Whebell S, Quick L, Luke S, et al. Effect of early adjunctive vasopressin initiation for septic shock patients: a target trial emulation. Crit Care. 2025;29(1):188.
36. Liu ZM, Chen J, Kou Q, Lin Q, Huang X, Tang Z, et al. Terlipressin versus norepinephrine as infusion in patients with septic shock: a multicentre, randomised, double-blinded trial. Intensive Care Med. 2018;44(11):1816–25.
37. Wu Z, Zhang S, Xu J, Xie J, Huang L, Huang Y, et al. Norepinephrine vs Vasopressin: Which Vasopressor Should Be Discontinued First in Septic Shock? A Meta-Analysis Shock. 2020;53(1):50–7.
38. Jeon K, Song JU, Chung CR, Yang JH, Suh GY. Incidence of hypotension according to the discontinuation order of vasopressors in the management of septic shock: a prospective randomized trial (DOVSS). Crit Care. 2018;22(1):131.
39. Mallmann C, Galiotto TMB, de Oliveira MS, Moraes RB. Reduction of norepinephrine versus vasopressin in the stabilization phase of septic shock: RENOVA clinical trial. Med Intensiva (Engl Ed). 2025:502147.
40. Kattan E, Ibarra-Estrada M, Jung C. Knowing the ropes of vasopressor dosing: a focus on norepinephrine. Intensive Care Med. 2024;50(4):587–9.

Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

BioMed Central publishes under the Creative Commons Attribution License (CCAL). Under the CCAL, authors retain copyright to the article but users are allowed to download, reprint, distribute and /or copy articles in BioMed Central journals, as long as the original work is properly cited.